detailed ophthalmological examination, performed by an ophthalmologist.

During the patient's stay in the hospital, Aralen was discontinued because of suspicion (later shown to be unfounded) that it might have caused the neuritis.

As the degree of disability forbade the patient's returning to work, the issue of her going to live in her son's home was forced. During the stay in hospital, administration of prednisolone, 10 mg. daily, was begun, and a week later when she left the hospital the patient was walking. However, the use of prednisolone had to be discontinued after three weeks because of gastric distress.

For the next two and a half years, the patient lived in her son's home doing light household tasks. There never again was overt evidence of the reactive depression which had been so apparent at the time of her entering the hospital. During these years results of repeated urinalysis, blood cell counts, and determination of sedimentation rate were always within normal range and the patient was never febrile. A neurological consultant who examined the patient in February, 1959, reported a complete recovery from the peripheral neuritis.. The discoid lupus of the face was also almost entirely cleared.

DISCUSSION

It cannot be proved that in this patient peripheral neuritis was caused by lupus erythematosus. However, this connection cannot be proved absolutely in any patient who has lupus and peripheral neuritis since the two conditions are only rarely associated and the pathological findings in those nerves which have been studied under the microscope in the reported cases are not uniquely different from those found in other collagen diseases. What sets the present case apart from the others that have been reported with this concurrence is the fact that the patient did not die.

An aspect of this case—and one usually not mentioned in reports of cases of lupus erythematosus—was the effect of emotionally charged events in the patient's life on the course of the lupus. In this patient, peripheral neuritis appeared after the husband died. The patient was an immigrant with no roots in the community in which she was living. The marriage of her only son and the death of her husband, in rapid succession, caused a reactive depression which chronologically was related to the appearance of neuritis. Whether or not it was the actual cause of neuritis is a question that cannot be answered. It should be noted in this connection that some investigators have expressed a belief that a reactive depression may usher in an attack of disseminated lupus erythematosus.[6]

When the patient went to live in her son's home, her depression abated, and later the neuritis and lupus cleared also, even though active medical treatment had been discontinued. Whether or not this change in the patient's life was partially responsible for the lessening of the organic diseases is another unanswerable question.

SUMMARY

In the present case peripheral polyneuritis occurred in association with discoid lupus erythematosus. This is the first case so far reported in the literature in which such an association has occurred. An attempt has been made in this report to touch upon a neglected subject in the literature of lupus erythematosus—namely, the influence of psychological factors on the disease in certain patients.

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Temporal Arteritis

A Report of Two Cases Without Systemic Symptoms

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TEMPORAL ARTERITIS is a rare disease that is being recognized with increasing frequency. It has usually been regarded as a localized disease of the temporal arteries,[6,9,11] but gradually the description of the disease has been expanded to include not only all the cranial arteries,[2,17] but almost any other artery in the body. It has been described as occurring in the coronary arteries[9,13] and in other larger arteries.[4]

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The clinical features of the disease were first described by Hutchinson in 1890,[10] but it was not until 1932 that the pathologic features were described, by Horton, Magath and Brown.[8]

This disease is invariably described as one accompanied by systemic symptoms of fever, chills, loss of weight, anemia, leukocytosis and accelerated erythrocyte sedimentation rate.[4,6,12,13] It occurs chiefly in women between the ages of 55 and 80.[2,8,13] It is extremely rare in Negroes.[9] The disease is self-limited, lasting from one to 20 months, the average being about 12 months.[3,13] The local features are described as consisting of bead-like, reddened temporal arteries which are very tender to touch, with excruciating headache over the involved area.* The cranial symptoms are usually described as lethargy, mental retardation, vertigo, dysarthria, delirium and painful jaws and mouth.[2,8,11] In about one-third of the cases there is ophthalmic involvement, such as diplopia, photophobia and visual field defects, and permanent blindness often occurs.[11,17]

Temporal arteritis is a disease of unknown cause, although it has been thought to be a hypersensitivity state which may be part of the systemic disease of periarteritis nodosa.[4] It has also been thought to be a bacterial disease, but repeated bacterial studies have been inconclusive.[9] Some authorities believe it to be merely a manifestation of arteriosclerosis, but the evidence is more in favor of a granulomatous or inflammatory process. The occurrence of a case in a woman, aged 25, would tend to discredit the arteriosclerotic concept.[14]

Microscopically the lesions of this disease are rather characteristic and give the impression of a generalized granulomatous and inflammatory process involving all the layers of the arterial wall.[3,12] The adventitia shows infiltration with neutrophils, lymphocytes, and rarely eosinophils. There is medial necrosis, and characteristic foreign body giant cells of the Langhans type are always found in the media. There is a fibrous tissue infiltration of the intima, and sometimes thrombi may be seen in the narrowed lumen of the vessel.[8,11,13] In the eye, the pathologic changes are those of ischemia or ischemic infarction of the optic nerve.[17]

Although the question has not yet been resolved with any degree of finality, evidence is accumulating that temporal arteritis is a systemic and generalized disease. It has been said that it is merely a localized manifestation of periarteritis nodosa,[2] but there is some convincing evidence against this concept. In periarteritis nodosa only the smaller arteries (3 mm. or 4 mm.) are involved; whereas in temporal arteritis the larger arteries are the site of the disease. There is no aneurysmal formation of the arteries in temporal arteritis as there is in periarteritis nodosa. Finally, the characteristic medial necrosis and giant cell formation which is always found in temporal arteritis is rarely, if ever, seen in periarteritis nodosa,[8,11,12] and furthermore in temporal arteritis the eosinophils are scarce in the artery wall. Clinically, the disease differs from periarteritis nodosa by its relatively benign course. It can not be said to differ clinically in its localization, for evidence is accumulating that the characteristic pathologic lesions, heretofore described only in the temporal arteries, may be found in many other arteries.[2,4,13]

The treatment of temporal arteritis was not too satisfactory until the advent of steroid therapy in 1950.[3,5,11,15] Until then, the most widely used treatment was either procaine infiltration about the involved vessel or excision of a portion of it. It was felt that the symptomatic improvement resulting from these measures was due to interruption of periarterial sensory fibers.[6,14] Many other treatments have been used but have not stood the test of time. These include the administration of antihistamines, antibiotics, iodides, sulfonamides, cobra venom, nicotinic acid, thiamine, mercury and local x-ray therapy.[7,15,16] A recent case report told of the successful use of phenylbutazone in therapy.[1]

The following two cases are being reported because they are thought to be of unusual interest in that in each case temporal arteritis was present in a fairly localized manner without any systemic symptoms whatever.

REPORTS OF CASES

CASE 1. A 66-year-old white woman was first observed in the office on February 12, 1959, with chief complaint of severe generalized headaches and "knots on the head" for one month. She also complained of some lacrimation in the right eye. She had been seen elsewhere and treated with Empirin and codeine with no relief. The headaches were becoming progressively worse, and would even wake her at night. She had had no loss of weight, no weakness or fever.

The patient had had a thyroidectomy three years previously for toxic goiter, an operation for herniated intervertebral disc in 1945 and exploratory laparotomy in 1922 and again in 1950, with no pathologic condition found in either instance.

Upon physical examination a decided fullness and beading of the temporal arteries, which were reddened and tender, was noted. The patient was obese. The temperature was 98.0° F. The blood pressure was 140/80 mm. of mercury.

Hemoglobin content was 12.7 gm. per 100 cc. Erythrocytes numbered 4.7 million per cu. mm., and the leukocyte count was 10,000 per cu. mm. with a differential of 68 per cent polymorphonuclears, 26 per cent lymphocytes, 1 per cent eosinophils and 5 per cent monocytes. The erythrocyte sedimentation rate was 54 mm. in one hour. An electrocardiogram was within normal limits. No abnormality was seen in an x-ray film of the chest. Results of urinalysis revealed no abnormalities.

*References 2, 5, 10, 11, 13.

Administration of dexamethasone was begun, 0.75 mg. three times a day, and the next day the patient had complete relief of headache. A biopsy specimen from the left temporal artery showed a pronounced inflammatory process involving all the layers of the artery. The adventitia was infiltrated with neutrophils and leukocytes and there were no eosinophils. Necrosis and giant cells were seen in the media, and there was decided proliferation of fibrous tissue of the intima.

The dose of dexamethasone was gradually reduced until the patient was receiving a maintenance dose of 0.75 mg. daily. When last seen on May 26, 1959, she was still asymptomatic, but the erythrocyte sedimentation rate still was 51 mm. in one hour.

CASE 2. A 70-year-old white woman was first seen in the office on May 11, 1959, with complaint of severe generalized headaches for two weeks. She had had a head cold with bilateral earache two weeks previously and had been treated with penicillin. She had been seen elsewhere and given codeine with no relief. The headaches became progressively worse. There were no complaints referrable to the eyes, and no weakness, fever, loss of weight or anorexia.

Hysterectomy for prolapsed uterus had been done six months previously. Otherwise the patient always had been in good health.

On examination the temporal arteries on both sides were observed to be distended and reddened and extremely tender to touch. Nodules could be palpated along the course of the vessels. The blood pressure was 150/80 mm. of mercury and the body temperature was 97.8° F.

Erythrocytes numbered 4.4 million per cu. mm. and the hemoglobin content was 12.0 gm. per 100 cc. Leukocytes numbered 10,400 per cu. mm.—73 per cent polymorphonuclears, 26 per cent lymphocytes and 1 per cent monocytes. The erythrocyte sedimentation rate was 54 mm. in one hour. An electrocardiogram was within normal limits. Results of urinalysis revealed no abnormalities.

A clinical diagnosis of temporal arteritis was made, and the patient was given dexamethasone, 0.75 mg. three times a day. By the following day, she had complete relief of the headache. Biopsy of a specimen of the right temporal artery was done and changes typical of temporal arteritis were seen —pronounced inflammatory involvement of all the layers of the vessel, with many neutrophils and lymphocytes in the adventitia; an occasional eosinophil; decided medial necrosis and foreign body cells of Langhans type; and fibrous tissue inflammation of the intima.

The dose of dexamethasone was gradually reduced to a maintenance level of 0.75 mg. daily. When last seen, on June 30, 1959, the patient was still completely asymptomatic, although the erythrocyte sedimentation rate was 50 mm. in one hour.

COMMENT

In each of the two cases the circumstances and symptoms were typical, as far as they went, of temporal arteritis. An elderly white woman with excruciating headaches unrelieved by strong analgesics, the temporal arteries bead-like, prominent and inflamed. Missing, however, were the anorexia, fever, loss of weight, leukocytosis and weakness described in so many of the some 350 cases reported in the literature. Each had, however, a persistently accelerated erythrocyte sedimentation rate, which, although in itself not necessarily indicative of systemic disease, is frequently a concomitant of localized inflammatory disease of many types.

Perhaps the early diagnosis and treatment in these cases restricted the disease process; or perhaps the early abatement of symptoms lends support to the idea that temporal arteritis may be a process localized to the temporal arteries only.

This report is also believed to be the first in which use of dexamethasone for temporal arteritis is described.

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